view. In particular, it appeared that a number of severely ill or moribund patients, who later died, had no instrumental monitoring during anaesthesia and that 3% of deaths occurred in low risk patients and 25% in moderate risk patients.

The level of routine monitoring suggested may be criticised because it is expensive of time and money or, as suggested by Dr A S L Lamb, because it causes a distraction (9 October, p 1043). Neither criticism is tenable. In the former case the application of a blood-pressure cuff is quick and costs little; disposable electrocardiogram electrodes may cost 50 pence for each patient, but an electrocardiogram mat is cheap (and can be homemade), non-disposable, rapidly applied, and gives an adequate signal; and a Wright respirometer costs less than a cheap ventilator alarm and can be used for both spontaneously breathing and mechanically ventilated patients. The second criticism, namely distraction, has been applied to electrocardiogram monitoring. Indeed, it would be if brought out only on high days and holidays. Only routine use of blood-pressure, electrocardiogram, and ventilatory monitoring will remove their novelty value and inform rather than distract. This state of affairs can be achieved only by routine monitoring of all patients including the fit. When the unexpected happens, as indeed it will, it will be recognised rapidly and appropriate action will be taken sooner rather than when damage has been done or when monitoring devices have been rescued from dusty cupboards.

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[1] Lunn JN, Mushin WW. *Mortality associated with anaesthesia*. London: Nuffield Provincial Hospitals Trust, 1982.

Smallpox vaccination

SIR,—Minerva appears to have been under a misapprehension when she wrote in the *BMJ* (9 October, p 1054): "Variola virus for vaccination against smallpox is now held in only three laboratories—in South Africa, the USSR, and the United States."

For many years the virus used for vaccination against smallpox has been vaccinia virus, and not variola virus, the causative agent of smallpox. The concern of the medical community in the United States of America with reports of adverse reactions to smallpox vaccinations used to treat herpes infections[1] relates to the use of vaccinia virus and not variola virus as implied by Minerva.

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[1] *World Health Organisation Weekly Epidemiology Record* 1982;**57**:291.

**Minerva should have known better.—ED, *BMJ*.

Medical communication: the old and the new

SIR,—Mr David Loshak (9 October, p 1050) writes that: "Boring journals eventually go out of business." I wonder if this is true, other than in the sense that "in the long run we are all dead." There are several controlled circulation (giveaway) journals that have been excessively boring and pretty trivial in their content for 10 or even 20 years but which show no signs of disappearing and have indeed grown fatter, presumably because the demand for pharmaceutical advertising space is insatiable.

I probably had more pieces published in *World Medicine* during Michael O'Donnell's editorship than anyone who was not a staff member or a regular columnist. I was not one of those who promised never to write for the magazine again, and I have not had any offer of a contribution refused by Mr Loshak. But I do not want to write for *World Medicine* now because it has become dull and unstimulating, so that I can seldom bother to read more than a few lines of any article or column.

Perhaps *World Medicine* will pick up under Mr Loshak and regain some of its influence, the sort of influence, for instance, that brought about the reform of the GMC with late assistance from the BMA and the *BMJ*. But even if it does not pick up I am sure it will never go out of business: the NHS will continue to pay for pharmaceutical advertising even if most of it goes straight from doctors' letter boxes into their bonfires.

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Drug interaction with coumarin derivative anticoagulants

SIR,—Dr A A Sharp's letter (11 September, p 737) sorts out the quinidine error and goes some way towards explaining the dichloralphenazone anomaly, but it says nothing at all about the other mistakes in the paper of the Standing Advisory Committee for Haematology of the Royal College of Pathologists (24 July, p 274).

What about the recommendation that distalgesic is safe, when in some cases it has clearly been shown to induce severe bleeding? And what about all the other errors in the drug lists? Let me just detail a few from the first list of drugs, which is headed "Drugs expected to potentiate oral anticoagulants" so your readers can get some idea of the sheer number of doubtful and erroneous statements.

The very well controlled studies of O'Reilly and Udall and Waris clearly show that quite large amounts of alcohol in patients with normal liver function have no effect whatsoever on prothrombin times. What evidence is there for the claim that alcohol may be expected to potentiate the oral anticoagulants? Apart from a paper in 1957 which described a clinically insignificant effect when chlorpromazine was given with nicoumalone, what good evidence is there for the claim that it affects prothrombin times? There was a single case report in 1975 of an interaction with ethacrynic acid which remains unconfirmed. What other evidence is there? Naproxen does not affect the response to anticoagulants according to the work of Slattery. What evidence is there that it does? Triclofos sodium causes a transient change in prothrombin times and the work of BCDSP indicates that such changes are clinically unimportant. What evidence is there that they are important? Neomycin has been shown not to affect prothrombin times, and other aminoglycosides are unlikely to do so unless dietary levels of vitamin K are very low. What evidence is there of an interaction?

So out of the first list of 29 drugs, seven of them are not expected to potentiate the oral anticoagulants. And what about the next list? I should be most interested to see what evidence there is for 13 out of the 37 drugs listed. The lists are shot through with many questionable and erroneous claims.

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ABC of Diabetes: treatment

SIR,—I have enjoyed the series of articles by Dr P J Watkins entitled "ABC of Diabetes." I wonder, however, if I may be permitted to make a small addition to the section on treatment with sulphonylureas (19 June, p 1853). Dr Watkins properly draws attention to the problems in giving some sulphonylureas to patients with renal failure, but he has omitted gliclazide in the list of drugs that are chiefly metabolised before excretion and which may be given relatively safely in these circumstances. Gliclazide is over 80% metabolised, and although 60%-70% of the metabolites are excreted in the urine these are without hypoglycaemic activity.[1]

Lagrue and Riveline[2] conducted a study of 56 patients with diabetic nephropathy, and in a subgroup of 22 patients with serum creatinine from 115 to 3536 μmol/l (1·3 to 40 mg/100 ml) only one hypoglycaemic episode occurred, when the patient's dose was increased. Some preliminary results from our laboratory (unpublished observations) show that the biological half life remains unchanged in patients with severe renal impairment, and clearance in these patients is slightly increased, presumably due to enzyme induction.

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[1] Campbell DB, Adriaessens P, Hopkins YW, Gordon B, Williams JRB. Pharmacokinetics and metabolism of gliclazide: a review. *International Congress Series of the Royal Society of Medicine* 1980;**20**: 71-82.

[2] Lagrue G, Riveline B. Effects of long-term administration of gliclazide on proteinuria and renal function in patients with diabetic nephropathy. *International Congress Series of the Royal Society of Medicine* 1980;**20**:219-24.

ABC of Diabetes: pregnancy

SIR,—While agreeing entirely with Dr P J Watkins's criteria for diagnosing gestational diabetes (11 September, p 717) we would emphasise that if a glucose tolerance test is indicated before the last trimester of pregnancy and is normal this does not preclude the subsequent development of gestational diabetes. Serial glucose tolerance tests should be performed if indications persist. This is illustrated by a patient recently treated by us who was gestationally diabetic in her first pregnancy and successfully treated by diet alone (preprandial blood glucose 6 mmol/l (11 mg/100 ml)). In her second pregnancy she presented at 16 weeks with glycosuria. A